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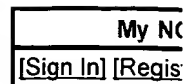
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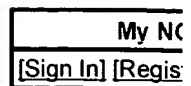
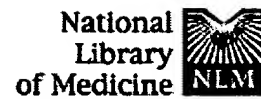
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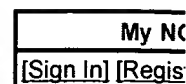
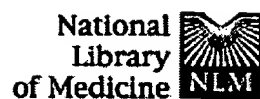
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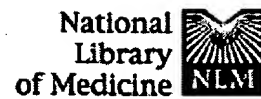
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**Monoclonal antibodies raised against a subsequence of senile plaque core protein react with plaque cores, plaque periphery and cerebrovascular amyloid in Alzheimer's disease.****Allsop D, Landon M, Kidd M, Lowe JS, Reynolds GP, Gardner A.**

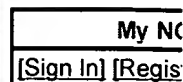
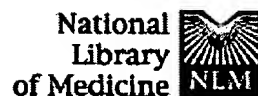
Four monoclonal antibodies (1D2/1/2, 1G10/2/3, 3B6/1/1, 4D12/2/6) were raised against a synthetic peptide consisting of residues 8-17 of a protein reported to be common to senile plaque cores, cerebrovascular amyloid and neurofibrillary tangles in Alzheimer's disease. In an immunoperoxidase study of Alzheimer brain tissue, these antibodies stained plaque and vascular amyloid but not tangles, suggesting that the polypeptide chain in the region of residues 8-17 is exposed in the former two but, if present, inaccessible in the latter. In addition, staining of granular material in the plaque periphery was observed. These antibodies will be useful tools for future work on the origin of this protein.

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### **A monoclonal antibody to amyloid in the brains of patients with Alzheimer's disease.**

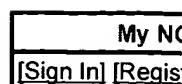
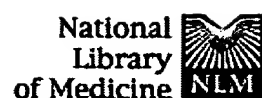
**Ishii T, Sato M, Haga S, Shinoda T, Kato K, Saito T.**

A monoclonal antibody that reacts with the amyloid in senile plaques in the brains of patients with Alzheimer's disease, was produced. The antigen was the crude amyloid protein (DAM, Pras et al. 1968) from the spleen of a patient with primary amyloidosis. The antibody crossreacts with DAM, but does not stain the amyloid of primary amyloidosis in tissue sections. The presence of a common antigenic site or sites in both the amyloid of senile plaques and amyloid of primary amyloidosis which react with this antibody is postulated.

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### Establishment of a monoclonal antibody against senile plaques and its application for immunohistological and immunoelectron microscopical studies in the brain of the elderly.

Kawasaki H, Utsuyama M, Takahashi H, Hayashi Y, Kurashima C, Esaki Y, Maruyama N, Hirokawa K.

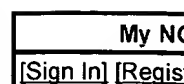
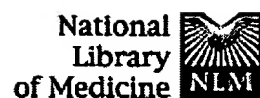
Department of Pathology, Yokufukai Geriatric Hospital, Tokyo, Japan.

A monoclonal antibody (Am-3) was produced against senile plaques in the brain of a patient with Alzheimer's disease. Am-3 was reactive with senile plaques of typical, primitive and diffuse type not only in the brain used as immunogen, but also those in the brain of 15 out of 25 autopsy cases of the aged people. Moreover, Am-3 was also reactive with granular materials of various sizes scattered in the 1st, 3rd and 4th layers of the cerebral cortices of the cases with severe dementia. Am-3 was also reactive with vessel wall of the congophilic angiopathy. By immunoelectron microscopic examination, Am-3 was positive with amyloid fibril in the core and crown of senile plaques, and in the congophilic angiopathy.

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1: Am J Pathol. 1989 May;134(5):973-8.

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**Monoclonal antibodies to a synthetic peptide homologous with the first 28 amino acids of Alzheimer's disease beta-protein recognize amyloid and diverse glial and neuronal cell types in the central nervous system.**

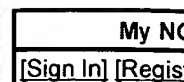
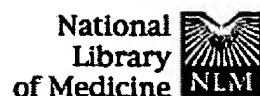
**Stern RA, Otvos L Jr, Trojanowski JQ, Lee VM.**

Department of Pathology and Laboratory Medicine (Neuropathology),  
University of Pennsylvania School of Medicine.

Studies were conducted to identify neural cells that synthesize and/or process cerebral amyloid using antisera and monoclonal antibodies (MAbs) raised to synthetic peptides based on the first 28 amino acids of the amyloid beta-protein. Using rabbit and mouse antisera, and 7 MAbs, sections of neocortex, hippocampus, cerebellum, and spinal cord from Alzheimer's disease (AD), Down's syndrome (DS), and control cases were probed. The antibodies produced 3 distinct immunohistochemical patterns: 1) staining restricted to neuritic plaque and blood vessel amyloid only (antisera, 1 of 7 MAbs); 2) immunoreactivity confined to cytoplasmic granules in diverse neuronal, glial (astrocytes, ependyma) and other (leptomeningeal, perivascular, choroid plexus) cells (1 of 7 MAbs); 3) a summation of these 2 patterns (5 of 7 MAbs). Controls resembled the AD and DS cases, except for a paucity of immunoreactive plaques and blood vessels in the controls. Immunoreactivity was reduced or removed by the peptides used to produce these antibodies. Formalin- and Bouins-fixed tissues reacted weakly or not at all with these antibodies while microwave denatured tissues reacted very intensely with them. Specific staining was enhanced by treatment of the tissue sections with Triton X-100, NaDodSO<sub>4</sub>, or trypsin. These studies significantly extend earlier studies that localized amyloid beta-protein precursor mRNA to human brain cells, and they suggest that the beta-protein, its precursor, and/or fragments thereof may exist in diverse neural cell types in AD, DS, and control brains.

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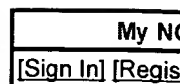
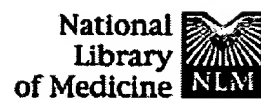
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Palo Alto Institute for Molecular Medicine, Mountain View, CA 94043.

The human monoclonal antibody (humAb) problem has largely been solved with the aid of the polymerase chain reaction (PCR) [Larrick et al., Bio/Technology 7 (1989a) 934-938; Larrick et al., Biochem. Biophys. Res. Commun. 160 (1989b) 1250-1256; Chiang et al., BioTechniques 7 (1989) 360-366]. Phage display has now made it possible to recover humAb with primary response level affinities (approx.  $10(6)$  M<sup>-1</sup>) for virtually any antigen (including self antigens) from comprehensive libraries of B-cell repertoires from non-immunized humans [Marks et al., J. Mol. Biol. 222 (1991) 581-597; Marks et al., Bio/Technology 10 (1992) 779-783; Griffiths et al., EMBO J. 12 (1993) 725-734]. This means that the goal of therapeutic humAb without immunization is within reach. However, in order to achieve the affinities generally required for therapeutic use ( $>$  or  $= 10(9)$  M<sup>-1</sup>), reliable methods will be needed to complete the affinity maturation process in vitro. Available X-ray crystallographic data and energy calculations indicate that only a fraction of the substantial contact surface between the Ab and protein antigens contribute significantly to affinity. Thus, the remaining contact surface presents multiple opportunities to develop additional high-affinity contacts, needing only a means to identify them. To this end, we have developed a computer-assisted method for oligodeoxyribonucleotide-directed scanning mutagenesis, called parsimonious mutagenesis (PM), whereby all three complementarity-determining regions (CDR) of a variable region (V-region) gene can be simultaneously and thoroughly searched for improved variants in libraries of manageable size. These libraries are made with low-redundancy 'doping' codons and biased nucleotide (nt) mixtures designed to maximize the abundance of combining sites with predetermined proportions of preselected sets of alternative amino acids (aa). This allows the library to 'probe' the surface of the antigen one or a few aa residues at a time with a wide selection of aa side chains to search out and identify new high-affinity contacts. In addition to affinity maturation in vitro, PM can also be used to remove unwanted cross-reactivities and to 'reshape' rodent mAb for human

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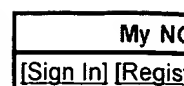
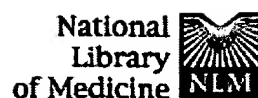
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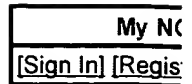
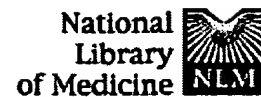
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Athena Neurosciences, Inc., South San Francisco, California 94080.

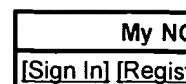
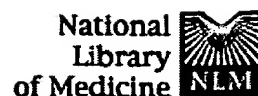
One of the major clinical findings in Alzheimer's disease (AD) is the formation of deposits of beta-amyloid protein in amyloid plaques, derived from the beta-amyloid precursor protein (beta-APP). To determine the possible use of beta-APP as a diagnostic marker for AD in CSF, a monoclonal antibody-based immunoassay specific for this protein was developed. The assay does not differentiate between beta-APP695 and beta-APP751 forms but does preferentially recognize beta-APP751 complexed with a protease. Of the two sets of CSF samples tested, one set, obtained from living patients, gave a slightly lower level of beta-APP in AD and Parkinson's disease patients relative to controls, whereas the other set, composed of postmortem samples, showed no significant differences between the AD and control groups.

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1: J Exp Med. 1993 Apr 1;177(4):1181-6.

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FREE full text article at  
www.jem.org**Human antibodies reactive with beta-amyloid protein in Alzheimer's disease.****Gaskin F, Finley J, Fang Q, Xu S, Fu SM.**

Department of Psychiatry, University of Virginia School of Medicine, Charlottesville 22908.

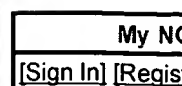
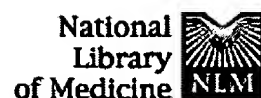
Four human B cell lines established by Epstein-Barr viral transformation of B cells from a patient with a clinical diagnosis of Alzheimer's disease (AD) were found to secrete antibodies that react with plaques and cerebrovascular blood vessels in AD brain in a staining profile characteristic of beta-amyloid protein (beta-AP) in AD brain. Two of these antibodies were shown to be reactive with a rare plaque in a normal brain. In these studies, immunofluorescence and avidin-biotin complex immunoperoxidase methodology were used to determine antibody reaction, and thioflavine S was used to double label amyloid and neurofibrillary tangles. The four antibodies also reacted with neurons in normal and AD brain. Absorption studies, dot immunoblots, and enzyme-linked immunosorbent assays with beta-amyloid peptides 1-28 (beta-A1-28) and 1-40 (beta-A1-40) indicate the major determinant of the reactive epitope is located in the region of amino acids 1-28 of beta-AP. However, inhibition studies demonstrate a significant contribution to the antigenic determinant by the 29-40 region of the beta-A1-40. These antibodies represent the first human autoantibodies against beta-AP. The pathological significance of these autoantibodies is discussed.

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☐ 1: Am J Pathol. 1994 May;144(5):1082-8.[Related Articles, Links](#)**Development of a monoclonal antibody specific for the COOH-terminal of beta-amyloid 1-42 and its immunohistochemical reactivity in Alzheimer's disease and related disorders.****Murphy GM Jr, Forno LS, Higgins L, Scardina JM, Eng LF, Cordell B.**Department of Psychiatry and Behavioral Sciences, Stanford University  
School of Medicine, California.

The beta-amyloid peptide (beta AP) has been characterized by protein sequencing techniques as a 39-43 amino acid protein with heterogeneous COOH-termini. Controversy exists regarding the predominant form of beta AP in neuritic plaques (NP) and cerebral vasculature of Alzheimer's disease (AD) brain. A monoclonal antibody was developed that selectively recognizes the free COOH-terminal of beta AP 1-42 but not beta AP species with shorter or longer COOH-termini. Brain sections from AD and related disorders were examined using this antibody. In AD samples, the antibody stained diffuse amyloid and NP cores, many intraneuronal and extraneuronal neurofibrillary tangles (NFT), but not cerebrovascular amyloid. Pick and Lewy bodies lacked immunoreactivity. These findings suggest that beta AP 1-42 is present in early and mature amyloid deposits and NFT, but that species of beta AP other than 1-42 comprise human vascular deposits.

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